

IN THE SPECIFICATION

Please replace Paragraph [0001] with the following:

[0001] This application is a divisional of copending U.S. Application No. 10/371,685 filed on 21 February 2003 which is a divisional of copending U. S. Application No. 09/890,559 filed on 01 August 2001 which was the National Stage of International Application No. PCT/US00/03022 filed on 03 February 2000 which claims the benefit of U.S. Provisional Application No. 60/119,038 which was filed on 05 February 1999

Please replace Paragraph [0012] with the following:

[0012] In one aspect, the present invention is a method of treating patients in need of treatment for a cardiac disorder, comprising administering to the patient a seven carbon fatty acid compound or derivative thereof, wherein the compound or derivative thereof is able to readily enter the mitochondrion without special transport enzymes. In a preferred method, the seven carbon fatty acid compound comprises n-heptanoic acid. In another preferred method, the seven carbon fatty acid compound comprises a triglyceride comprising n-heptanoic acid, for example, triheptanoin. In a preferred method, the derivative is a five carbon fatty acid chain. In another preferred method, the derivative is selected from the group consisting of 4-methylhexanoate, 4-methylhexenoate, 3-hydroxy-4-methylhexanoate, 5-methylhexanoate, 5-methylhexenoate and 3-hydroxy-5-methylhexanoate. In a preferred method, the compound or derivative thereof is capable of being broken down by normal β -oxidation in humans to methylbutyric acid. In another preferred method, the compound or derivative thereof is capable of being broken down by normal β -oxidation in humans to isovaleric acid. In another preferred method, the compound or derivative is capable of being broken down by normal β -oxidation in humans to n-valeryl-CoA. In yet another preferred method, the compound or derivative is capable of being broken down by normal β -oxidation in humans to propionyl-CoA in one or more oxidative procedures. Preferably, the compound or derivative thereof is provided to the patient in an amount comprising at least about 25% of the dietary caloric requirement for the patient. Preferably, the compound or derivative is provided parenterally or orally ~~orally, parenterally, or~~

~~intraperitoneally~~. This method is suitable for treating cardiac disorders such as cardiac myopathy. It is also suitable for treatment of the aftermath of heart surgery, wherein the compound or derivative is utilized for direct fueling of heart muscle.

Please replace Paragraph [0073] with the following:

[0073] Triheptanoin can be obtained by the esterification of heptanoic acid and glycerol by any means known in the art. Triheptanoin is also commercially available through Condea Chemie GmbH (Witten, Germany), now Sasol (Witten, Germany) as Special Oil 107.

Please replace Paragraph [0075] with the following:

[0075] The seven-carbon triglycerides of the present invention can be administered orally[[,]] or parenterally, ~~or intraperitoneally~~. Preferably, it can be administered via ingestion of a food substance containing a seven-carbon fatty acid source such as triheptanoin at a concentration effective to achieve therapeutic levels. Alternatively, it can be administered as a capsule or entrapped in liposomes, in solution or suspension, alone or in combination with other nutrients, additional sweetening and/or flavoring agents. Capsules and tablets can be coated with sugar, shellac and other enteric agents as is known.

Please replace Paragraph [0079] with the following:

[0079] Since propionyl-CoA is a metabolic by-product of triheptanoin oxidation, increased blood levels of propionic acid can result. Moreover, propionyl-CoA can enter into other enzymatic reactions which produce toxic compounds affecting the Krebs's cycle and the urea cycle. Therefore, the administration of a seven-carbon fatty acid such as n-heptanoic acid and/or triheptanoin supplement, especially in patients exhibiting a build-up of serum propionic acid, may require the administration of a carnitine supplement and/or a biotin and vitamin B12 combination. In the presence of excess L-carnitine and the mitochondrial enzyme carnitine acetyltransferase, propionyl-CoA is converted to propionylcarnitine, a non-toxic substance which is excreted in the urine. Biotin is a vitamin cofactor required for the enzyme propionyl-CoA carboxylase which catalyzes the conversion of propionyl-CoA to methylmalonyl-CoA. Cyanocobalamin is a form of vitamin B12 which acts as a cofactor for the enzyme methylmalonyl-CoA mutase which catalyzes the conversion of methylmalonyl-CoA to succinyl-

CoA. Succinyl-CoA is readily pulled into the Krebs's cycle. Therefore, excess propionyl-CoA in the patient's blood is removed by conversion to succinyl-CoA.